IN THE CLAIMS

1. (canceled)

2. (currently amended) A method of screening for therapeutic agents useful in the treatment of heart failure in a mammal comprising the steps of

i) determining an ~~the~~ activity of a formyl peptide receptor-like 2 (FPRL2) polypeptide in the absence of a test compound,

ii) determining the activity of said polypeptide in the presence of the test compound; ~~and~~

iii) identifying the test compound as a potential therapeutic agent for the treatment of heart failure if the activity of the FPRL2 polypeptide in the presence of the test compound is different than the activity of the FPRL2 polypeptide in the absence of the test compound; and

iv) determining whether the test compound has an effect on a symptom of heart failure in an *in vivo* assay,

wherein the FPRL2 polypeptide comprises the amino acid sequence SEQ ID NO:2 or an amino acid sequence which is at least 95% homologous to SEQ ID NO:2 and wherein the activity of the FPRL2 is an activity of a G protein coupled receptor.

3-26. (canceled)

27. (currently amended) The method of claim 32 ~~26~~, wherein the cell is *in vitro*.

28. (previously presented) The method of claim 2, wherein the FPRL2 polypeptide is in a cell-free system.

29-31. (canceled)

32. (previously presented) The method of claim 2, wherein the FPRL2 polypeptide is in a cell.

33. (previously presented) The method of claim 2 wherein the FPRL2 polypeptide comprises an amino acid sequence which is at least 95% homologous to SEQ ID NO:2.

34. (previously presented) The method of claim 2 wherein the FPRL2 polypeptide comprises an amino acid sequence which is at least 98% homologous to SEQ ID NO:2.

35. (previously presented) The method of claim 2 wherein the FPRL2 polypeptide comprises an amino acid sequence which is at least 99% homologous to SEQ ID NO:2.

36. (previously presented) The method of claim 2 wherein the FPRL2 polypeptide comprises the amino acid sequence SEQ ID NO:2.

37. (new) The method of claim 2 wherein the activity of the FPRL2 polypeptide is measured by an alteration in intracellular calcium concentration.